

L5 136 L4 AND NICOTINIC ACETYLCHOLINE
RECEPTOR/AB,BI

=> s l5 and promoter/ab,bi

'AB' IS NOT A VALID FIELD CODE
L6 27 L5 AND PROMOTER/AB,BI

=> dup rem l6

PROCESSING COMPLETED FOR L6
L7 11 DUP REM L6 (16 DUPLICATES REMOVED)

=> d l1- bib ab

YOU HAVE REQUESTED DATA FROM 11 ANSWERS -
CONTINUE? Y(N)/y

L7 ANSWER 1 OF 11 BIOSIS COPYRIGHT 2000 BIOSIS
DUPLICATE 1

AN 1999:222992 BIOSIS
DN PREV199900222992

TI Induction of utrophin gene expression by heregulin in skeletal
muscle

cells: Role of the N-box motif and G4 binding protein.

AU Gramolini, Anthony O.; Angus, Lindsay M.; Schaeffer, Laurent;
Burton,

Edward A.; Tinsley, Jonathan M.; Davies, Key E.;

Changeux,

*** Jean-Pierre*** ; Jasmin, Bernard J. (1)

CS (1) Department of Cellular and Molecular Medicine, Faculty of
Medicine,

University of Ottawa, 451 Smyth Road, Ottawa, ON, K1H 8M5

Canada

SO Proceedings of the National Academy of Sciences of the United
States of

America, (March 16, 1999) Vol. 96, No. 6, pp. 3223-3227.

ISSN: 0027-8424.

DT Article

LA English

SL English

AB The modulation of utrophin gene expression in muscle by the
nerve-derived

factor agrin plausibly involves the trophic factor ARIA/hergulin.

Here we

show that heregulin treatment of mouse and human cultured

myotubes caused

a approxq2.5-fold increase in utrophin mRNA levels. Transient

transfection

experiments with utrophin ***promoter*** -reporter gene

constructs

showed that this increase resulted from an enhanced transcription of
the

utrophin gene. In the case of the ***nicotinic***

acetylcholine ***receptor*** delta and epsilon

subunit genes,

E6 1 PICCIOTTO R/AU
E7 3 PICCIOTTO ROBERT/AU
E8 2 PICCIOTTO S/AU
E9 2 PICCIOTTO SONDRA/AU
E10 1 PICCIOTTO TED/AU
E11 3 PICCIOTTOC L/AU
E12 1 PICCIOTTOR G/AU

=> s e2-e5

L2 72 ('PICCIOTTO MARIAN R'/AU OR 'PICCIOTTO
MARINA'/AU OR 'PICCIOTTO
MARINA R'/AU OR 'PICCIOTTO MARINA
RACHEL'/AU)

=> e bassis alain/au

E1 1 BASSIRL RAD H/AU
E2 1 BASSIROU N/AU
E3 0 -> BASSIS ALAIN/AU
E4 1 BASSIS EFM/AU
E5 2 BASSIS L M/AU
E6 13 BASSIS M L/AU
E7 1 BASSISSI E/AU
E8 2 BASSISSI FABIO/AU
E9 24 BASSISSI P/AU
E10 1 BASSISSI PAOLA/AU
E11 9 BASSIST L/AU
E12 2 BASSIST L F/AU

=> e bessis alain/au

E1 52 BESSIS A/AU
E2 8 BESSIS A S/AU
E3 21 -> BESSIS ALAIN/AU
E4 1 BESSIS ANDRE/AU
E5 5 BESSIS ANNE SOPHIE/AU
E6 1 BESSIS C/AU
E7 89 BESSIS D/AU
E8 6 BESSIS DANIEL/AU
E9 3 BESSIS DIDIER/AU
E10 1 BESSIS E/AU
E11 11 BESSIS F/AU
E12 21 BESSIS G/AU

=> s e1-e3

L3 81 ('BESSIS A'/AU OR 'BESSIS A S'/AU OR 'BESSIS
ALAIN'/AU)

=> s l1 or l2 or l3

L4 514 L1 OR L2 OR L3

=> s l4 and nicotinic acetylcholine receptor/ab,bi

'AB' IS NOT A VALID FIELD CODE

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E1 1 CHANGEUX JEAN PIERRE/AU
E2 407 CHANGEUX JEAN PIERRE/AU
E3 0 -> CHANGEUX JEAN-PIERRE/AU
E4 1 CHANGEUX M J/AU
E5 2 CHANGEUX P/AU
E6 8 CHANGEUX T/AU
E7 1 CHANGEUX THOAMS/AU
E8 4 CHANGEUX NICOLAS/AU
E9 5 CHANGEZ M/AU
E10 1 CHANGEZ MOHAMMED/AU
E11 1 CHANGEZ MOHD/AU
E12 1 CHANGEZI AL TAMASH/AU

=> s e1-e2

L1 408 ('CHANGEUX JEAN PIERRE'/AU OR 'CHANGEUX
JEAN PIERRE'/AU)

=> e picciotto marina/au

E1 94 PICCIOTTO M R/AU
E2 1 PICCIOTTO MARIAN R/AU
E3 13 -> PICCIOTTO MARINA/AU
E4 57 PICCIOTTO MARINA R/AU
E5 1 PICCIOTTO MARINA RACHEL/AU

herregulin was previously reported to stimulate transcription via a conserved ***promoter*** element, the N-box, which binds the multimetric Ets-related transcription factor GA binding protein (GABP). Accordingly, site-directed mutagenesis of a single N-box motif in the utrophin gene ***promoter*** abolished the transcriptional response to herregulin. In addition, over-expression of herregulin, or of the two GABP subunits in cultured myotubes, caused an N-box-dependent increase of the utrophin ***promoter*** activity. In vivo, direct gene transfer into muscle confirmed that herregulin regulates utrophin gene expression. Finally, electrophoretic mobility shift assays and supershift experiments performed with muscle extracts revealed that the N-box of the utrophin ***promoter*** binds GABP. These findings suggest that the sub synaptic activation of transcription by herregulin via the N-box motif and GABP are conserved among genes expressed at the neuromuscular junction. Because utrophin can functionally compensate for the lack of dystrophin, the elucidation of the molecular mechanisms regulating utrophin gene expression may ultimately lead to therapies based on utrophin expression throughout the muscle fibers of Duchenne muscular dystrophy patients.

L7 ANSWER 2 OF 11 BIOSIS COPYRIGHT 2000 BIOSIS
 DUPLICATE 2
 AN 1998:307358 BIOSIS
 DN PREV199800307358
 TI Nonmyogenic factors bind ***nicotinic***
 acetylcholine
 receptor ***promoter*** elements required for response to denervation.
 AU Bessereau, Jean-Louis; Laudembach, Vincent; Le Poupon, Chantal.
 CS (1) Neurobiol. Mol., UA CNRS D1284, Dep. Biotechnol., Inst. Pasteur 25/28 rue du Dr. Roux, 75724 Paris Cedex 13 France
 SO Journal of Biological Chemistry, (May 22, 1998) Vol. 273, No. 21, pp. 12786-12793.
 ISSN: 0021-9258.
 DT Article
 LA English
 AB Nicotinic acetylcholine receptors (AChRs) belong to a class of muscle proteins whose expression is regulated by muscle electrical activity.

In innervated muscle fiber, AChR genes are transcriptionally repressed outside of the synapse, while after denervation they become reexpressed throughout the fiber. The myogenic determination factors (MDFs) of the MyoD family have been shown to play a central role in this innervation-dependent regulation. In the chicken AChR alpha-subunit gene ***promoter***, two E-boxes that bind MDFs are necessary to achieve the enhancement of transcription following muscle denervation. However, the deletion of ***promoter*** sequences located upstream to these E-boxes greatly impairs the response to denervation (Bessereau, J. L., Stratford-Perricaudet, L. D., Piette, J., Le Poupon, C. and P. (1994) Proc. Natl. Acad. Sci. U.S.A. 91, 1304-1308). Here we identified two additional cis-regulatory elements of the alpha-subunit gene ***promoter*** that cooperate with the E-boxes in the denervation response. One region binds the Sp1 and Sp3 zinc finger transcription factors. The second region binds at least three distinct factors, among which we identified an upstream stimulatory factor, a b-ZIP-HLH transcription factor. We propose that among MDF-responsive muscle promoters, a specific combination between myogenic and nonmyogenic factors specify innervation-dependent versus innervation-independent promoters.

L7 ANSWER 3 OF 11 BIOSIS COPYRIGHT 2000 BIOSIS
 DUPLICATE 3
 AN 1998:318654 BIOSIS
 DN PREV199800318654
 TI Implication of a multisubunit Ets-related transcription factor in synaptic expression of the ***nicotinic*** ***acetylcholine*** ***receptor***.
 AU Schaeffer, Laurent; Duclert, Nathalie; Huchet-Dymannus, Monique.
 CS (1) CNRS UA D1284 'Neurobiologie Moléculaire', Inst. Pasteur, 25 rue du Dr. Roux, F-75724 Paris Cedex 13 France
 SO EMBO (European Molecular Biology Organization) Journal, (June 1, 1998) Vol. 17, No. 11, pp. 3078-3090.
 ISSN: 0261-4189.
 DT Article
 LA English
 AB In adult muscle, transcription of the ***nicotinic***

acetylcholine ***receptor*** (AChR) is restricted to the nuclei located at the neuromuscular junction. The N-box, a new ***promoter*** element, was identified recently and shown to contribute to this compartmentalized synaptic expression of the AChR delta- and epsilon-subunits. We demonstrate that the N-box mediates transcriptional activation in cultured myotubes and identify the transcription factor that binds to the N-box as a heterooligomer in myotubes and adult muscle. The GABP (GA-binding protein) alpha-subunit belongs to the Ets family of transcription factors, whereas the beta-subunit shares homology with IkappaB and Drosophila Notch protein. GABP binding specificity to mutated N-box in vitro strictly parallels the sequence requirement for beta-galactosidase targeting to the endplate in vivo. In situ hybridization studies reveal that the mRNAs of both GABP subunits are abundant in mouse diaphragm, with preferential expression of the alpha-subunit at motor endplates. In addition, herregulin increases GABPalpha protein levels and regulates phosphorylation of both subunits in cultured chick myotubes. Finally, dominant-negative mutants of either GABPalpha or GABPbeta block herregulin-elicited transcriptional activation of the AChR delta and epsilon genes. These findings establish the expected connection with a presynaptic trophic factor whose release contributes to the accumulation of AChR subunit mRNAs at the motor endplate.

L7 ANSWER 4 OF 11 BIOSIS COPYRIGHT 2000 BIOSIS
 AN 1999:13173 BIOSIS
 DN PREV199900013173
 TI ***Promoter*** analysis of the neuronal ***nicotinic*** ***acetylcholine*** ***receptor*** alpha4 gene: Methylation and expression of the transgene.
 AU Watanabe, Hajime; Zoli, Michele; ***Changeux, Jean-Pierre (1)***
 CS (1) Neurobiol. Mol., CNRS URA 1284, Inst. Pasteur, 25-28 rue du Dr. Roux, 75724 Paris Cedex 13 France
 SO European Journal of Neuroscience, (July, 1998) Vol. 10, No. 7, pp. 2244-2253.
 ISSN: 0953-816X.
 DT Article
 LA English
 AB Neuronal ***nicotinic*** ***acetylcholine*** ***receptor***

(nAChR) subunit genes compose a family of genes. The major isoform of nAChR in the brain is made up of the alpha4 and beta2 subunits and possesses a high affinity for nicotine. To investigate the mechanisms of the regulation of the nAChR alpha4 gene expression in mouse, its genomic DNA was cloned and characterized. The transcription initiation site was mapped by primer extension and RNase protection experiments and localized at about 254 bp upstream of the translation initiation site. The 5' flanking region of this gene did not have typical TATA box but GC-rich sequences were found around the initiation site. Methylation analysis of this region revealed that genomic DNAs from liver and muscle are partially methylated, whereas little methylation was observed in genomic DNA from brain. To characterize the cis-acting elements driving cell-specific expression of the alpha4 subunit gene, we produced lines of transgenic mice which carry a series of fragments of the alpha4 gene fused with bacterial lacZ as a reporter gene. An 11.5-kb DNA fragment containing 9 kb of the region upstream of the transcription initiation site and the intron was found to confer an expression pattern which coincides rather well with the endogenous gene expression pattern at early embryonic stages, suggesting that the elements necessary for the onset of alpha4 gene expression are located in this region. A DNA fragment containing the 1.8-kb upstream sequence and the first intron drove expression of lacZ in a limited subset of alpha4 expressing cells, whereas the 1.8-kb upstream sequence alone did not elicit any significant expression. These results show that both upstream and intronic sequences are important for cell-specific expression of the nAChR alpha4 gene.

L7 ANSWER 5 OF 11 INPADOC COPYRIGHT 2000 EPO

LEVEL 1

AN 13786614 INPADOC

TI ***PROMOTER*** OF BETA2-SUBUNIT OF NEURONAL ***NICOTINIC*** ***ACETYLCHOLINE*** ***RECEPTOR***, TRANSGENIC ANIMALS

IN CHANGEUX, JEAN-PIERRE; PICCIOTTO, MARINA; BESSIS, ALAIN

INS ***CHANGEUX JEAN-PIERRE*** ; ***PICCIOTTO MARINA*** ; ***BESSIS***

PA INSTITUT PASTEUR

PAS PASTEUR INSTITUT

DT Patent

PIT NZA COMP. SPECIFICATION ACCEPTED

PI NZ 280674 A 19970922

AI NZ 1995-280674 A 19951214

PRAI US 1994-358627 A 19941214

L7 ANSWER 6 OF 11 MEDLINE DUPLICATE 4

AN 97303230 MEDLINE

DN 97303230

TI The neuron-restrictive silencer element: a dual enhancer/silencer crucial for patterned expression of a nicotinic receptor gene in the brain.

AU ***Bessis A*** ; Champiaux N; Chatelin L; Changeux J P

CS Neurobiologie Moleculaire, UA CNRS D1284, Departement des Biotechnologies, Institut Pasteur 25/28 rue du Dr Roux, 75724 Paris Cedex 15, France..

abisses@pasteur.fr

SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1997 May 27) 94 (11) 5906-11.

Journal code: PV3, ISSN: 0027-8424.

CY United States

DT Journal, Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals; Cancer Journals

EM 199708

EW 19970803

AB The neuron-restrictive silencer element (NRSE) has been identified in several neuronal genes and confers neuron specificity by silencing transcription in nonneuronal cells. NRSE is present in the ***promoter*** of the neuronal ***nicotinic*** ***acetylcholine*** ***receptor*** beta2-subunit gene that determines its neuron-specific expression in the nervous system. Using transgenic mice, we show that NRSE may either silence or enhance transcription depending on the cellular context within the nervous system. In vitro in neuronal cells, NRSE activates transcription of synthetic promoters when located downstream in the 5' untranslated region, or at less than 50 bp upstream from the TATA box, but switches to a silencer when located further upstream. In contrast, in nonneuronal cells NRSE always functions as a silencer. Antisense RNA inhibition shows that the NRSE-binding protein REST contributes to the activation of transcription in neuronal cells.

L7 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2000 ACS

AN 1996:422699 CAPLUS

DN 125:78571

TI Genomic DNA fragments coding for the .beta.2-subunit of the neuronal ***nicotinic*** ***acetylcholine*** ***receptor*** and its transfection into transgenic animals

IN ***Changeux, Jean-Pierre*** ; ***Picciotto, Marina*** ; ***Bessis,***

*** Alain***

PA Institut Pasteur, Fr.

SO Eur. Pat. Appl., 36 pp.

CODEN: EPXXDW

DT Patent

LA English

FAN CNT 1

PATENT NO. KIND DATE APPLICATION NO. DATE

PI EP 717105 A2 19960619 EP 1995-402872 19951214

EP 717105 A3 19990407

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE

CA 2163098 AA 19960615 CA 1995-2165098 19951213

AU 9540373 A1 19960620 AU 1995-40373 19951213

JP 08242866 A2 19960924 JP 1995-326094 19951214

PRAI US 1994-358627 19941214

AB Several genes encoding subunits of the neuronal nicotinic acetylcholine receptors have been cloned and regulatory elements involved in the transcription of the .alpha.2 and .alpha.7-subunit genes have been described. Yet, the detailed mechanisms governing the neuron-specific transcription and the spatio-temporal expression pattern of these genes remain largely uninvestigated. The .beta.2-subunit is the most widely expressed neuronal nicotinic receptors subunit in the nervous system. The structural and regulatory properties of the 5' sequence of this gene were studied. A 1163-bp fragment of upstream sequence is sufficient to drive the cell-specific transcription of a reporter gene in both transient transfection assays and in transgenic mice. Deletion anal. and site-directed mutagenesis of this ***promoter*** reveal 2 neg. pos. element. The pos. acting sequence includes one functional E-box. One of the repressor elements is located in the transcribed region and is the NRSE/RE1 sequence already described in promoters of neuronal genes. The regulatory elements from the .beta.2-subunit-encoding sequences can be used to direct the neuron-specific expression of a nucleotide sequences

encoding a reporter, tumorigenic, oncogenic, or immortalizing foreign proteins.

L7 ANSWER 8 OF 11 MEDLINE DUPLICATE 5
AN 96313792 MEDLINE
DN 96313792
TI Differential regulation of neuronal ***nicotinic***
acetylcholine ***receptor*** subunit gene promoters by Bm-3
POU family transcription factors.
AU Millon N G; ***Bessis A***; Changeux J P; Lachman D S
CS Department of Molecular Pathology, University College London Medical School, UK.
SO BIOCHEMICAL JOURNAL, (1996 Jul 15) 317 (Pt 2) 419-23.
Journal code: 9YO. ISSN: 0264-6021.
CY ENGLAND; United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199611
AB The regulatory region of the neuronal nicotinic acetylcholine (nACh) receptor alpha 2 subunit gene is activated by the Bm-3b POU transcription factor but not by the closely related factors Bm-3a and Bm-3c. This pattern of regulation has not previously been observed for other neuronally expressed genes, several of which, such as those encoding alpha-interneuron or SNAP-25, are activated by Bm-3a and Bm-3c but repressed by Bm-3b. The alpha 3 nACh receptor subunit gene is also shown to be activated by Bm-3a but is repressed by Bm-3b and Bm-3c. In contrast, the Bm-3 POU family transcription factors have no effects on either the alpha 7 or beta 4 nACh receptor subunit genes. The Bm-3b on the alpha 2 subunit are thus in contrast to the inhibitory actions of Bm-3b on several promoters that are activated by Bm-3 alpha.
The different actions of the Bm-3 POU factors on the range of nACh receptor genes tested suggests that the novel stimulation of the alpha 2 subunit by Bm-3b is specific to this subunit and not a general feature of nACh receptor genes.

L7 ANSWER 9 OF 11 MEDLINE DUPLICATE 6
AN 95318076 MEDLINE
DN 95318076
TI The neuronal ***nicotinic*** ***acetylcholine*** ***receptor*** alpha 2 subunit gene ***promoter*** is activated by the Bm-3b

POU family transcription factor and not by Bm-3a or Bm-3c.
AU Millon N G; ***Bessis A***; Changeux J P; Lachman D S
CS Department of Molecular Pathology, University College London Medical School, UK.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1995 Jun 23) 270 (25) 15143-7.
Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199510
AB The regulatory region of the neuronal ***nicotinic*** ***acetylcholine*** ***receptor*** alpha 2 subunit gene, which contains six copies of the octamer-related sequence CCCATGCCAAT, is activated by the Bm-3b POU family transcription factor but not by the closely related factors Bm-3a and Bm-3c. This effect is in contrast to the previously documented inhibitory effect of Bm-3b on octamer-containing promoters that are activated by Bm-3a and Bm-3c.
Activation of the alpha 2 gene by Bm-3b requires that both the POU domain and other N-terminal sequences are derived from Bm-3b and is dependent on the inactivity of the alpha 2 gene regulatory region, being lost in truncated derivatives containing one, two, or four copies of the octamer-related sequence. Surprisingly, however, these truncated derivatives are activated by Bm-3c. These effects are discussed in terms of both the influence of the target sequence and its context in the ***promoter*** on activation by the various forms of Bm-3 as well as of the processes that restrict expression of the alpha 2 subunit gene to a few cells in the nervous system.

L7 ANSWER 10 OF 11 MEDLINE DUPLICATE 7
AN 96164254 MEDLINE
DN 96164254
TI ***Promoter*** elements conferring neuron-specific expression of the beta 2-subunit of the neuronal ***nicotinic*** ***acetylcholine*** ***receptor*** studied in vitro and in transgenic mice.
AU ***Bessis A***; Salmon A M; Zoli M; Le Nov'ere N; Picot M;
Changeux J P
CS UA CNRS D1284, Departement des Biotechnologies, Institut Pasteur 25/28, Paris, France.

SO NEUROSCIENCE, (1995 Dec) 69 (3) 807-19.
Journal code: NZR. ISSN: 0306-4522.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-X82655; GENBANK-M31433; GENBANK-M90489; GENBANK-M53301; GENBANK-X63509; GENBANK-L11891; GENBANK-X17102
EM 199606
AB Several genes encoding subunits of the neuronal nicotinic acetylcholine receptors have been cloned and regulatory elements involved in the transcription of the alpha 2 and alpha 7-subunit genes have been described. Yet, the detailed mechanisms governing the neuron-specific transcription and the spatio-temporal expression pattern of these genes remain largely uninvestigated. The beta 2-subunit is the most widely expressed neuronal nicotinic receptor subunit in the nervous system. We have studied the structural and regulatory properties of the 5' sequence of this gene. A fragment of 1163 bp of upstream sequence is sufficient to drive the cell-specific transcription of a reporter gene in both transfection assays and in transgenic mice. Deletion analysis and site-directed mutagenesis of this ***promoter*** reveal two negative elements and one positive element. The positively-acting sequence includes one functional E-box. One of the repressor elements is located in the transcribed region and is the NRSE/RE1 sequence already described in promoters of neuronal genes. In this paper, we describe the neuron-specific ***promoter*** of the gene encoding the neuronal ***nicotinic*** ***acetylcholine*** ***receptor*** beta 2-subunit.

L7 ANSWER 11 OF 11 MEDLINE DUPLICATE 8
AN 93275748 MEDLINE
DN 93275748
TI Negative regulatory elements upstream of a novel exon of the neuronal ***nicotinic*** ***acetylcholine*** ***receptor*** alpha 2 subunit gene.
AU ***Bessis A***; Savatier N; Devillers-Thierry A; Bejanin S; Changeux J P
CS UA CNRS D1284, Departement des Biotechnologies, Institut Pasteur, Paris,

France.
SO NUCLEIC ACIDS RESEARCH, (1993 May 11) 21 (9) 2185-92.

Journal code: O8L ISSN: 0305-1048.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
OS GENBANK:X69511
EM 199309
AB The expression of the ***nicotinic*** ***acetylcholine***
receptor alpha 2 subunit gene is highly restricted to the
Spiriform lateralis nucleus of the Chick diencephalon. As a first
step
toward understanding the molecular mechanism underlying this
regulation,
we have investigated the structural and regulatory properties of the
5'
sequence of this gene. A strategy based on the ligation of an
oligonucleotide to the first strand of the cDNA (SLIC) followed by
PCR
amplification was used. A new exon was found approximately 3kb
upstream
from the first coding exon, and multiple transcription start sites of
the
gene were mapped. Analysis of the flanking region shows many
consensus
sequences for the binding of nuclear proteins, suggesting that the 1
kb
flanking region contains at least a portion of the ***promoter***
of
the gene. We have analysed the negative regulatory elements
present within
this region and found that a silencer region located between
nucleotide
-144 and +76 is active in fibroblasts as well as in neurons. This
silencer
is composed of six tandem repeat Oct-like motifs
(OCCCATGCAAT), but does
not bind any member of the Oct family. Moreover these motifs
were found to
act as a silencer only when they were tandemly repeated. When
two, four or
five motifs were deleted, the silencer activity of the motifs
unexpectedly
became an enhancer activity in all cells we have tested.

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E CHANGEUX JEAN-PIERRE/AU

L1 408 S E1-E2
E PICCOTTO MARINA/AU
L2 72 S E2-E5
E BASSIS ALAIN/AU
E BASSIS ALAIN/AU
L3 81 S E1-E3
L4 514 S L1 OR L2 OR L3
L5 136 S L4 AND NICOTINIC ACETYLCHOLINE
RECEPTOR/AB,BI
L6 27 S L5 AND PROMOTER/AB,BI
L7 11 DUP REM L6 (16 DUPLICATES REMOVED)
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